

Magnesium in human physiology and pathology

(Magnez w fizjologii i patologii człowieka)

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Abstract – The paper discusses the biochemical and physiological significance of magnesium. Also, attention is paid to the development of lesions related to magnesium deficiency.

Key words - magnesium, biochemistry, physiology, lesions.

Streszczenie – Autorzy przedstawili znaczenie biochemiczne i fizjologiczne magnezu. Zwrócili także uwagę na rozwój zmian chorobowych związanych z niedoborami magnezu.

Słowa kluczowe - magnez, biochemia, fizjologia, zmiany chorobowe.

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Authors' contributions to the article:

- A. The idea and the planning of the study
- B. Gathering and listing data
- C. The data analysis and interpretation
- D. Writing the article
- E. Critical review of the article
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I. THE BIOCHEMICAL SIGNIFICANCE OF MAGNESIUM

Magnesium is an important element of organisms. Its content in respective tissues in human organism is presented in Table 1.

Table 1. The content of magnesium in selected human tissues [1]

The magnesium content in:	Amount	Unit
bone tissue	700-800	ppm
muscle tissue	900	ppm
erythrocytes	1,65-2,65	mmol/l
serum	0,65-1,05	mmol/l
hair tissue	40-120	ppm
the organism (total)	20-35	g

The regulative activity of magnesium is basically four-fold:

The participation in energy transformations

Magnesium is an element required for the synthesis of different compounds with energy-rich bonds, such as ATP (adenosine triphosphate), GTP (guanosine triphosphate), UTP (uridine triphosphate), CTP (cytidine triphosphate), ITP (inosine triphosphate), with a guanidinium-phosphate bond in phosphocreatine, enol phosphate bond of phosphoenol pyruvic acid, acetyl phosphate bond of 1,3-diphosphoglycerate and the bond of thioester acyl-coenzyme A or succinyl-CoA [2-4].

Magnesium is also essential to the use of energy-rich bonds, which is the case when they undergo hydrolysis or a phosphoryl group is transferred [5].

The role in the respiratory chain

Magnesium is responsible for the harmony of phosphorylation processes linked to the chain of oxidation-reduction processes. The participation of this bioelement in oxidation-reduction reactions is related to the formation of phosphopyridine nucleotides: DPN (diphosphopyridine nucleotide), TPN (triphosphopyridine nucleotide), flavin nucleotides: fla-

vin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) as well as the formation of coenzyme A [3,4,6].

The participation in the synthesis and activity of enzymes

For some enzymes, magnesium can be their integral component (e.g. alkaline phosphatase). For most of them, however, the bioelement is unstably bonded with an enzyme activator. Magnesium ions stimulate over 300 metabolic reactions by means of altering the spatial configuration of enzyme active centres [2,3,5].

One of the biologically relevant effects of magnesium activity is the activation of the enzymes related to a transformation in proteins and nucleic acids, such as transferase (nuclear transferase), synthetase and kinase.

The nuclear transferase of RNA participates in RNA (predominantly tRNA) synthesis. This enzyme, coupled with polyribosome initiation factors and factors prolonging peptide chain as well as amino acid-coding polyamines, contributes to the formation of proteins, whereas nucleotide transferase in DNA allows one to recreate and recombine the DNA [4].

The regulation of the functioning of cell membranes and intracellular organelles

Magnesium, by means of chelate bonds with cell and organelle membrane lipids, has impact on their functioning, facilitating the transportation of biological substance.

The bond between magnesium and phospholipids of cell membranes decreases their fluidity and permeability, while parallel electrostatic polarisation effects take place. In case of magnesium deficiency, the permeability of cell membranes increases, which leads to electrolyte shifts: the increase of intracellular concentration of Ca^{2+} and Na^{+} as well as the decrease in the K^{+} and phosphor concentration [3,6,7].

The changes in the permeability of cell membranes for electrolytes caused by the changing magnesium concentration are also relevant for the functioning of cell membrane receptors which participate, among others, in neutral transmission [8,9]. An example of that are NMDA glutamate receptors (stimulated N-Methyl-D-aspartic acid - NMDA). One of the most important characteristics of this receptor is the dependence on the block potential on the part of magnesium ions (Figure. I).

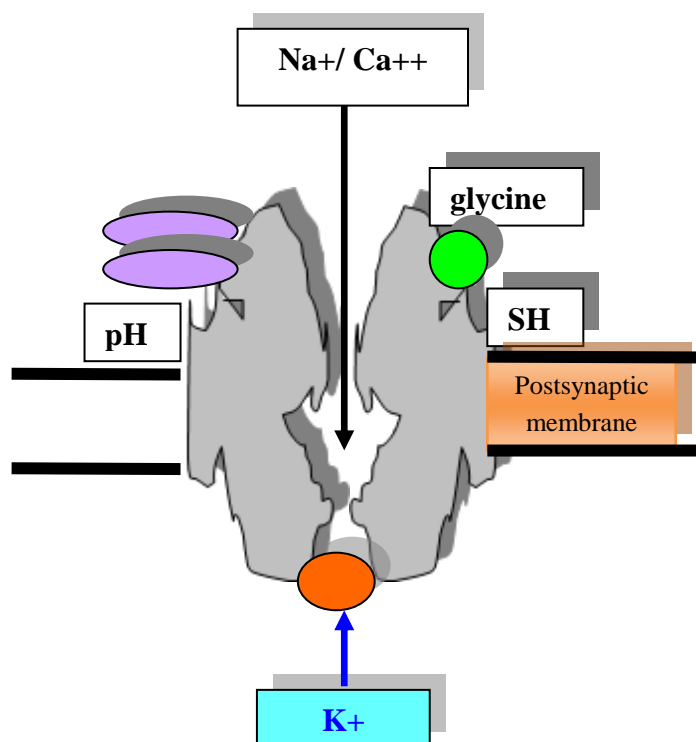
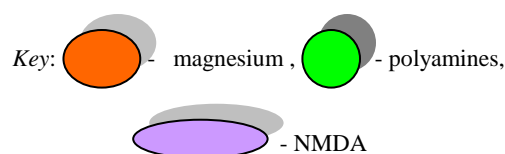


Figure I. NMDA receptor diagram



II. THE AETIOLOGY OF MAGNESIUM DEFICIENCY

Magnesium deficiency is always a result of negative balance, which may be caused by [10,11]:

- An insufficient supply of magnesium in food
- Insufficient absorption of magnesium from the digestive tract caused by, for instance, intestine inflammation or the loss of magnesium in bile, gastric or intestinal juice (biliary or intestinal fistulas)
- Excessive magnesium loss in urine caused by a primary kidney disease (primary tubulopathy with magnesium loss, Bartter syndrome, tubular acidosis, drug-induced tubulopathy)
- Excessive build-up of magnesium in bones (in patients who had a parathyroid adenoma removed) or in tissues (in cases of acute pancreatitis), the movement of magnesium to cells (in acute patients treated with insulin), the loss of magnesium in sweat or milk.

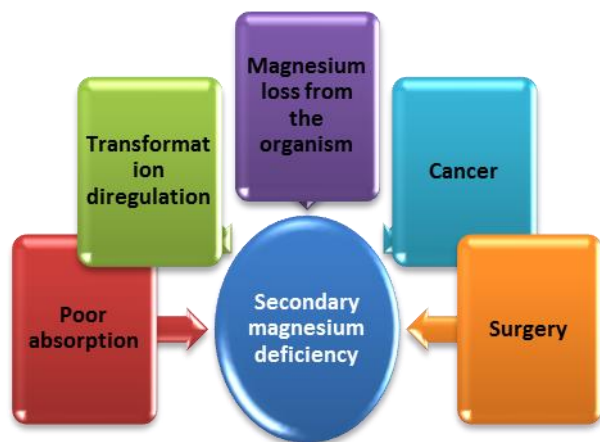


Figure II . The aetiology of secondary magnesium deficiency [3,6,7,11]

The significance of magnesium for the neuromuscular system

The biochemical processes dependent on magnesium provide the foundations for the functioning of all the organism's organs and systems. However, for some of them – like the neuromuscular system – the bioelement is of crucial significance. Magnesium deficiency causes limb and tongue tremor, myoclonus, Chvostek and Trousseau signs, tetany, muscle weakness, paraesthesia, apathy, depression, hallucinations, tremor, dizziness, nystagmus and sleepiness [1,12,13,14].

Magnesium deficiency also yield metabolic and cardiovascular symptoms such as: hypocalcaemia insensitive to calcium supplementation or sensitive to magnesium, hypokalaemia insensitive or hardly sensitive to magnesium, hypersensitivity to cardiac glycosides, ventricular cardiac arrhythmia, arterial hypertension or coronary insufficiency. Furthermore, magnesium deficiency is believed to have impact on the pathogenesis of nephrolithiasis, anaemia, functional disorders of digestive tract and osteomalacia [10,11].

III. THE PATHOPHYSIOLOGY OF MAGNESIUM DEFICIENCY SYMPTOMS

The cause of neuromuscular symptoms of magnesium deficiency is the intensified acetylcholine release in neural plates, which increases the excitability of myocytes and nerve cells. The increase in the Ca^{2+} influx to cells and the decrease of K^+ and Mg^{2+} ions in those cells lead to the disorders of the resting potential and repolarisation of cell membrane. These disorders are a cause of hypersensitivity to cardiac glycosides the occurrence of dangerous cardiac arrhythmia. Magnesium deficiency may also contribute to arterial hypertension (as the Ca^{2+} influx

to vascular myocytes is increased), coronary insufficiency (increased constriction of coronary vessels) and myocardial infarction (the deficiency destabilises the thrombocyte membrane and causes intensified thrombocyte aggregation). Magnesium deficiency may also cause hypercholesterolaemia and increased urine lithogenicity. What is more, it may contribute to kaliuresis that is resistant to the supplementation of potassium, which leads to hypovolaemia [10,11,14].

IV. REFERENCES

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